Arterial Stiffness and Autonomic Modulation After Free-Weight Resistance Exercises in Resistance Trained Individuals

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Abstract

Kingsley, JD, Mayo, X, Tai, YL, and Fennell, C. Arterial stiffness and autonomic modulation after free-weight resistance exercises in resistance trained individuals. *J Strength Cond Res* 30(12): 3373–3380, 2016–We investigated the effects of an acute bout of free-weight, whole-body resistance exercise consisting of the squat, bench press, and deadlift on arterial stiffness and cardiac autonomic modulation in 16 (aged 23 ± 3 years; mean ± *SD*) resistance-trained individuals. Arterial stiffness, autonomic modulation, and baroreflex sensitivity (BRS) were assessed at rest and after 3 sets of 10 repetitions at 75% 1-repetition maximum on each exercise with 2 minutes of rest between sets and exercises. Arterial stiffness was analyzed using carotid-femoral pulse wave velocity (cf-PWV). Linear heart rate variability (log transformed [ln] absolute and normalized units [nu] of low-frequency [LF] and high-frequency [HF] power) and nonlinear heart rate complexity (Sample Entropy [SampEn], Lempel-Ziv Entropy [LZEn]) were measured to determine autonomic modulation. BRS was measured by the sequence method. A 2 × 2 repeated measures analysis of variance (ANOVA) was used to analyze time (rest, recovery) across condition (acute resistance exercise, control). There were significant increases in cf-PWV ($p = 0.05$), heart rate ($p = 0.0001$), normalized LF (LFnu; $p = 0.001$), and the LF/HF ratio ($p = 0.0001$). Interactions were also noted for ln HF ($p = 0.006$), HFnu ($p = 0.0001$), SampEn ($p = 0.001$), LZEn ($p = 0.005$), and BRS ($p = 0.0001$) such that they significantly decreased during recovery from the resistance exercise compared with rest and the control. There was no effect on ln total power, or ln LF. These data suggest that a bout of resistance exercise using free-weights increases arterial stiffness and reduces vagal activity and BRS in comparison with a control session. Vagal tone may not be fully recovered up to 30 minutes after a resistance exercise bout.

Key Words heart rate variability, heart rate complexity, strength exercise, baroreflex sensitivity

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Introduction

Resistance training offers significant improvements in muscle and bone mass and is currently recommended by health organizations as a means to prevent and improve a host of chronic diseases (13). Although the effects of aerobic exercise on cardiovasculature control are well known, the responses to resistance exercise are less clear (18,27,29). An acute bout of resistance exercise may cause a short-time increase in central arterial stiffness (7,14,38); however, this is not a universal finding (17,19,22). In addition, numerous investigators have demonstrated that an acute bout of resistance exercise elicits a transient reduction of vagal modulation measured by several parameters of heart rate variability (HRV) and/or complexity (HRC) (18,20,21,31) and decreases in baroreflex sensitivity (BRS) (14).

Most protocols that have evaluated the effect of resistance exercise on vascular (8,19) and cardiac (18,20,21,31) control in healthy individuals were designed using solely, or mainly, weight machines, with a few exceptions (4,10,22). Therefore, the effects after an acute bout of whole-body free-weight resistance exercises on cardiovasculature control should be elucidated. This is important because cardiovascular responses to free-weights may differ in comparison with weight machines since free-weights may have a higher muscular activation in main (9), agonist (25) and/or stabilizing muscles (33). This higher muscle mass involved may lead to a transient increase in central arterial stiffness (11) modulated by increased sympathetic neural activation after exercise (34). In this sense, the higher metaboreflex activation due to an increased glycolytic involvement during the resistance exercise provoked by the higher muscle mass activation may cause a greater withdrawal of vagal modulation, and increased sympathetic activity, after the resistance exercise session (28) resulting in an increase in arterial stiffness (26,35). This increase in arterial stiffness then leads to decreased BRS (26). The difference in metabolic demand would result in different responses in terms of arterial stiffness, autonomic modulation, and BRS, making comparing weight machines with free-weights complex. To the best of our knowledge, no study has evaluated the effect of free-weight whole-body

resistance exercise on arterial stiffness and BRS in resistance-trained individuals. Also, only one study has evaluated autonomic modulation in response to free-weight whole-body resistance exercise in young, resistance-trained individuals (4). In that study, nationally and internationally competitive weightlifters performed back squat, seated shoulder press, deadlift, and front squat with intensities between 60% and 95% 1-repetition maximum (1RM), finding that parasympathetic activity returned to baseline values by 72 hours. Although this study highlights the effect of free-weight exercises on the cardiovasculature, it does it in a very specific population such that it is not necessarily generalizable to those individuals who use these whole-body free-weight resistance exercises as part of their training regime. It is clear that more research is needed to explain the effects of whole-body free-weight resistance exercise on vascular and cardiac modulation in resistance-trained individuals.

Therefore, the purpose of the present study was to examine the alterations in vascular and cardiac modulation after a period of heavy resistance exercise using whole-body free-weights in resistance-trained individuals. We hypothesized that there would be significant increases in central arterial stiffness primarily mediated by reductions in vagal modulation and BRS after the acute bout of whole-body free-weight resistance exercise.

Methods

Experimental Approach to the Problem

Measurements were made in each participant at rest and after the acute bout of resistance exercise or a quiet control. For all resting data collection, participants arrived to the laboratory had abstained from food for 3 hours, caffeine and alcohol for 12 hours, and strenuous exercise for 24 hours. On arrival to the laboratory participants rested in the supine position for 10 minutes before any data collection began. Carotid-femoral pulse wave velocity (cf-PWV) was collected for minutes 10 to 15, followed by autonomic and baroreflex measurements. Once these data were collected participants completed either the acute bout of resistance exercise or the quiet control. Within 2 minutes of completion of the acute bout of resistance exercise participants returned to the supine position for recovery data collection. The quiet control consisted of participants lying in the supine position for 30 minutes to match the time spent in the acute resistance exercise condition. During recovery, carotid-femoral pulse wave velocity (cf-PWV) was collected after a 10-minutes rest period and autonomic and baroreflex measurements were collected from minute 25 to 30.

Subjects

Based on our previous study investigating changes in Sample Entropy (SampEn) after an acute bout of whole-body resistance exercise, an effect size of 1.2 was calculated (21). For a power of 80%, a minimum of 15 participants were needed. Sixteen participants (men = 11, women = 5) that had been participating in resistance training for at least 3 days a week for a minimum of 2 years volunteered for the study. All participants were free from cardiovascular, musculoskeletal, metabolic disease, were nonsmokers, nonhypertensive, and were classified as nonobese. All participants also reported, through a questionnaire, that they were not taking any supplements or prescription medications that might alter vascular or autonomic function. All women underwent testing during the follicular phase of their menstrual cycle as noted by the start of their period. All participants signed an informed consent before the collection of any data having been made aware of the benefits and risks of the study. This project was approved by the Kent State University Institutional Review Board and conformed to the Declaration of Helsinki.

Anthropometrics

Height and weight were measured using a stadiometer and a balance beam scale, respectively. Height was measured to the nearest 0.1 cm. Weight was measured to the nearest 0.1 lb and converted to kg. Body mass index was reported with kg/m^2. Body composition was assessed with the 7-site skinfold analysis using published criteria (12). The Brozek equation was used to determine body density (3).

Maximal Strength

Maximal strength was assessed in the Strength Laboratory with the 1RM test using published standards (2). In essence, each participant lifted the maximal amount of weight that they could through a full range of motion with proper form and technique for the squat, bench press, and deadlift. Two minutes of rest was given between attempts and exercises. The squat and bench press were supervised by strength and conditioning specialists. The deadlift occurred on an Olympic platform. Seventy-two hours after the initial 1RM, participants returned to the Strength Laboratory for verification of their 1RM. The highest weight lifted over the 2 days was recorded as the maximal strength and was used to prescribe the workload for the acute exercise session and data analysis.

Pulse Wave Velocity

All measurements were collected following the guidelines of the Clinical Application of Arterial Stiffness, Task Force III (36). Before collection of cf-PWV, blood pressure was assessed with the SphygomoCor (AtCor Medical, Sydney, Australia) device to calibrate the pressure wave using an oscillometric cuff. Two separate blood pressures were taken, and they had to be within ± 5 mm Hg of each other. If the variability was greater than that, a third measurement was taken. The data were recorded as mean arterial pressure (MAP). A high-fidelity tonometer (SPT-301B; Millar Instruments, Houston, TX, USA) was used to obtain the pressure waveform from the right common carotid artery and the right femoral artery. The distances from the site of carotid sampling to the femoral artery and the carotid artery to the suprasternal notch were measured using a straight

line through a tape measure. The distance from the carotid artery to the suprasternal notch was then subtracted from the carotid–femoral segment length by the SphygmoCor system. The foot of the pressure wave was identified using an intrinsic algorithm of the software that detects the initial upstroke through a tangent line to the initial systolic upstroke point of the pressure tracing and an intersecting horizontal line through the minimum point (6). Chiu et al. (1991) demonstrated that this algorithm is very reproducible. The time delay between a minimum of 10 simultaneously recorded pressure flow waves was averaged by the software. Cf-PWV was essentially calculated from the distance between measurement points and measured time delay between proximal and distal foot waveforms using the following equation: PWV $= D/\Delta t$ in which D is the distance in meters and Δt is the time interval in seconds. In the present study, values obtained are indicative of aortic stiffness. The SphygmoCor software only uses those cf-PWV values that have a standard deviation of $\leq$10%. This technique has previously been shown to be highly reproducible (37). Reliability of cf-PWV in our laboratory was based on preliminary data using 6 young, healthy individuals at rest, that were 3 hours postprandial on 4 separate days. Each measurement was separated by at least 72 hours. Each measurement was made in duplicate, and then subsequently averaged for data analysis. The intraclass correlation coefficient for cf-PWV in our laboratory was 0.94.

Autonomic Modulation

Continuous 3-lead electrocardiogram (ECG) and beat-by-beat blood pressure recording signals were collected using a modified CM5 configuration (PowerLab; AD Instruments, Colorado Springs, CO, USA) and from the right middle finger using a ccNexfin (BMEYE, Amsterdam, The Netherlands), respectively. ECG was collected at a rate of 1000 Hz and blood pressure was obtained with a sampling frequency of 200 Hz. ECG recordings were used to determine heart rate (HR) at rest and during recovery. During all autonomic data collection a metronome was set at 12 breaths per minute.

Both of the signals were imported into WinCPRS (Absolute Aliens, Turku, Finland) after inspection of noise, ectopics, and artifacts. Fast Fourier transformation was used to generate spectral power. HRV was assessed in both the frequency and time domains. For the frequency domain, total power of HRV was used as a global index of autonomic modulation. Low-frequency power (LF; 0.04–0.15 Hz) is composed of both parasympathetic and sympathetic components. High-frequency power (HF; 0.15–0.4 Hz) was used as a measure of parasympathetic modulation. The LF/HF ratio was used as a measure of sympathovagal balance. The power spectra were calculated in both absolute (ms^2) and normalized units (nu) to evaluate the power of each component in relation to the total power. Normalized units (LF or HF) are determined by the division of the total power with the removal of the very low-frequency power ($<$0.04 Hz) by the power of a component and then multiplying by 100. Normalized measures of LF (LFnu) are indicative of sympathetic modulation, whereas normalized measures of HF (HFnu) are used as a measure of parasympathetic modulation.

SampEn and Lempel-Ziv entropy (LZEn) were used as methods for examining the RR interval complexity over the 5-minute epoch after removal of the linear trend. SampEn has been defined as the probability of matches or sequences being similar over a short period of time, which has a range of 0–2 (32). The more predictive the signal, the closer the value is to 0; the more chaotic, the closer to 2 (32).

LZEn was calculated using the same data points that were used to determine SampEn. This particular measure, which is based on Kolmogorov estimates, determines the number of different and repeating patterns, from short to long, and produces a chain of binary code. When the value is above the mean, the code "1" is inserted, when below the mean, the code "0" is inserted. From this binary code, LZEn is obtained and is similar to SampEn such that a value near 0 is interpreted as a fixed signal, whereas higher values are translated as more chaotic.

We calculated BRS using the sequence method of the RR interval and systolic blood pressure (30). The sequence method involves 3 or more consecutive beats that are denoted by increases or decreases of systolic blood pressure and RR interval of their following beat (Lag 1) changed in the same direction. The threshold change was set at 5 milliseconds for RR interval and 1 mm Hg for systolic blood pressure. Only sequences with correlations $\geq$0.80 were accepted. All data were collected following previously reported recommendations (30).

Acute Bout of Resistance Exercise

The acute bout of resistance exercise consisted of 75% 1RM with 3 sets of 10 repetitions for the squat, bench press, and deadlift. Two minutes of rest was given between sets and exercises. Strength and conditioning specialists supervised each session and provided a spotter if needed. Each bout of resistance exercise was approximately 30 minutes.

Table 1. Physical and functional characteristics of the participants ($N = 16$).*

	Participants
Age (y)	23 ± 3
Height (m)	1.74 ± 0.11
Weight (kg)	82.2 ± 17.7
Body fat (%)	16.5 ± 7.7
Fat mass (kg)	13.6 ± 8.2
Lean mass (kg)	68.5 ± 13.1
Squat (kg)	144 ± 32
Bench press (kg)	109 ± 33
Deadlift (kg)	160 ± 40

*Data displayed as means ± *SD*.

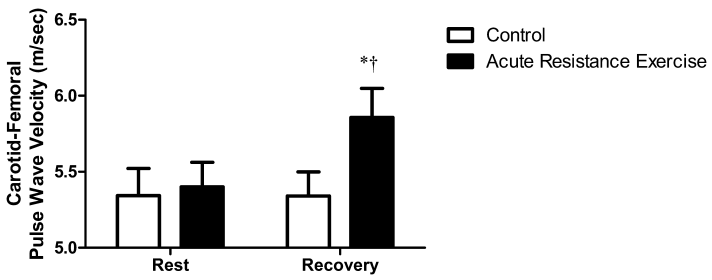

Figure 1. Change in carotid-femoral pulse wave velocity measured at rest and during recovery from a control and an acute bout of whole-body free-weight resistance exercise in young, resistance-trained individuals ($N = 16$). Data are mean ± *SEM*. *$p \leq 0.05$, significantly different from rest. †$p \leq 0.05$, significantly different from control.

Statistical Analyses

A one-way analysis of variance (ANOVA) was used to determine differences in all resting variables between the 2 days of testing. A Kolmogorov–Smirnov test revealed that total power, absolute LF, and absolute HF were not normally distributed. These variables were subsequently logarithmically transformed (ln). A 2 × 2 repeated measures ANOVA was used to determine the effects of time (rest and recovery) across condition (acute resistance exercise and control) on cf-PWV, MAP, HR, ln total power, ln LF, ln HF, LF/HF, HFnu, LFnu, SampEn, LZEn, and BRS. If the ANOVA noted a significant interaction, paired *t*-tests were used for post hoc comparisons. Significance was set *apriori* at $p \leq 0.05$. Values are presented as mean ± standard deviation (*SD*) except in figures which are presented as mean ± standard error of the mean (*SEM*). All statistical analyses were completed using IBM SPSS version 21 (Amrok, NY, USA).

Results

Participant characteristics are presented in Table 1. There was a significant interaction ($p = 0.05$) for cf-PWV such that it increased by 9.6% after the acute bout of resistance exercise compared with no change in the control (Figure 1). There was no significant change in MAP in response to the acute bout of resistance exercise (Table 2).

There was a significant interaction for heart rate (+59%, $p < 0.0001$; Table 2) such that it was significantly elevated after the acute bout of resistance exercise and compared with the control. There were no significant interactions for ln total power (−7.8%, $p = 0.09$) and ln LF (−1.4%, $p = 0.35$) after the acute bout of resistance exercise (Table 2). There was also a significant interaction for ln HF (−26.9%, $p = 0.006$) such that it decreased after the bout of resistance exercise but not the control (Table 2). There was a significant interaction for HFnu such that it significantly decreased (−59%, $p < 0.0001$) during recovery from the acute resistance exercise compared with rest and to the control. LFnu significantly increased (+92%, $p < 0.001$) above rest during recovery from the acute resistance exercise and compared with the control. In addition, there was a significant interaction for the LF/HF ratio (+624%, $p < 0.0001$; Table 2) such that it was elevated above the control and rest during the recovery. There was also a significant interaction ($p \leq 0.001$) for SampEn such that it decreased (−35.2%) during

Table 2. Autonomic variables at rest and during recovery from a control session and an acute bout of whole-body free-weight resistance exercise in young, resistance-trained individuals ($N = 16$).*†

	Control		Acute resistance exercise	
	Rest	Recovery	Rest	Recovery
Mean arterial pressure (mm Hg)	84 ± 5	86 ± 6	85 ± 5	88 ± 6
Heart rate (bpm)	60 ± 11	56 ± 10	58 ± 9	89 ± 10‡§
ln total power (ms^2)	8.20 ± 0.97	8.89 ± 1.40	8.19 ± 1.08	7.56 ± 3.01
ln LF (ms^2)	6.65 ± 0.98	7.80 ± 1.80	6.59 ± 1.13	6.50 ± 3.21
ln HF (ms^2)	7.40 ± 1.34	7.91 ± 1.37	7.13 ± 1.32	5.22 ± 3.70‡§
LF/HF	0.75 ± 0.90	0.85 ± 0.90	0.75 ± 0.61	5.4 ± 4.2‡§
Sample entropy	1.39 ± 0.32	1.62 ± 0.36	1.55 ± 0.28	1.04 ± 0.36‡§
Lempel-Ziv entropy	0.79 ± 0.16	0.88 ± 0.10	0.83 ± 0.013	0.66 ± 0.22‡§

*HF = High-frequency power; LF = Low-frequency power; LF/HF = Low-frequency power/High-frequency power ratio; ln = natural log function.
†Results are mean ± *SD*.
‡Significantly different from rest ($p \leq 0.05$).
§Significant group by time interaction ($p \leq 0.05$).

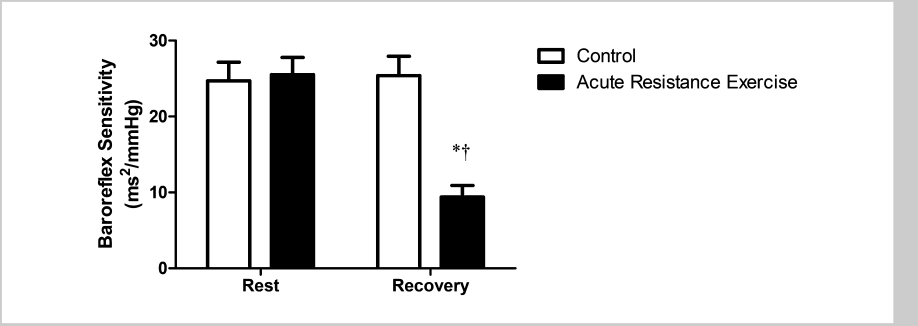

Figure 2. Change in baroreflex sensitivity measured at rest and during recovery from a control or an acute bout of whole-body free-weight resistance exercise in young, resistance-trained individuals ($N = 16$). Data are mean ± *SEM*. *$p \leq 0.05$, significantly different from rest. †$p \leq 0.05$, significantly different from control.

recovery from the resistance exercise bout and increased (+10.7%) during recovery from the control (Table 2). There was a significant interaction ($p = 0.005$) for LZEn such that it decreased by 20.5% after the acute bout of resistance exercise and increased by 12.1% from the control (Table 2). There was a significant interaction ($p = 0.0001$) for BRS such that it decreased by 63.1% after the acute bout of resistance exercise and didn't change from the control (Figure 2).

Discussion

The present study sought to elucidate changes in arterial stiffness, autonomic modulation, and baroreflex sensitivity in response to an acute bout of whole-body free-weight resistance exercise using free-weights. The primary findings of the present study are that after an acute bout of whole-body free-weight resistance exercise there are (a) increases in arterial stiffness, (b) decreases in vagal modulation, and (c) decreases in baroreflex sensitivity.

Our data suggest that whole-body free-weight resistance exercise using free-weights significantly increases arterial stiffness. The significant increase of 9.6% in cf-PWV in our study agrees with some of the reports that have demonstrated a transient increase in arterial stiffness in response to acute resistance exercise (7,14,38). Heffernan et al. (2007a) observed a significant increase of 20.8% from baseline to 20 minutes after the acute bout of resistance exercise when participants underwent 3 sets with the 10RM for 8 exercises. With the same resistance exercise protocol, Collier et al. (2010) observed a significant increase of 9.8% in cf-PWV from baseline to 40 minutes and 60 minutes after the session. Yoon et al. (2010) observed an increase of 2% in cf-PWV after 20 minutes in comparison with a control session that was not significantly different at 40 minutes after the resistance exercise. In this particular study, participants performed 2 sets of 15 repetitions at 60% 1RM for 8 exercises. Differences in reaching muscular failure (7) or a higher relative load used (38) in our work in comparison with the preceding studies may justify the differences in the increased values of central arterial stiffness. The difference between the work by Heffernan et al. (2007a) and Collier et al. (2010) was the time that the measurement was assessed. Our data, measured at 15 minutes after the acute bout of resistance exercise, had an increase in cf-PWV similar to the work by Collier et al. (2010) at 40 and 60 minutes. The data from the present study, and these previous studies, suggest that volume may be more important in terms of increasing arterial stiffness than just intensity. However, our results differ from others that have reported no differences in central arterial stiffness after resistance exercise (16,19,22). However, these studies involved the utilization of either lower- (17,19) or upper-body exercises only (22). These differences in cf-PWV in comparison with our study may be due to the total amount of muscle mass involved in the resistance exercise session. Protocols with a whole-body design produced a significantly increased cf-PWV (7,14,38). This increased central arterial stiffness can be originated by the inclusion of whole-body free-weight exercises, since the amount of muscle mass involved in resistance exercise determines the sympathetic activity (34), leading to the distensibility of the arteries (11).

The present study further demonstrates that an acute bout of resistance exercise reduces vagal modulation compared with rest. The significant decrease of 59% in HFnu in our study supports previous studies. Collectively, the data demonstrate that an acute bout of resistance exercise decreases vagal modulation in young healthy individuals (18,20,21,31), whether the participants are resistance-trained or not (21) up to 30 minutes. A descriptive analysis of the data suggests that in comparison with protocols using a similar total volume (i.e., number of exercises × number of sets), the reduction in vagal modulation is up to two-fold higher with whole-body free-weight resistance exercise in comparison with weight machines resistance exercise protocols (59% vs. 7.3–32.4%, respectively) (20,21). With our whole-body free-weight design, the reduction of vagal modulation was even higher in comparison with protocols performed with machines with a total volume of exercises between 6 and 10 (i.e., 59% vs. 12.4–25.2%) (18,31), which is a novel finding. A candidate for the increased vagal withdrawal in our free-weight protocol in comparison with other protocols with weight machines is the raised glycolytic involvement during the resistance exercise provoked by

the higher metaboreflex activation due to a large participation of the main (9), agonist (25), and/or stabilizing muscles (33). In turn, this may provoke a higher parasympathetic withdrawal (28). The high training status of the participants seems to have no role in the loss of vagal modulation since a previous study observed that the loss in variability is not affected by the experience condition of the individual in resistance training (21). It is worth noting that differences in body positions may account for some differences in the quantity of loss of vagal control. In the present study, data were collected in the supine position compared with previous studies that were performed in the seated position (20,31). This is important because body position affects the vagal activity at rest due to orthostatic load, with higher values in the supine position in comparison with seated position (23).

This is the first study to exhibit significant decreases in HRC after an acute bout of whole-body free-weight resistance exercise in resistance-trained individuals. The significant decrease in SampEn of 35.2% indicates a loss of complexity in the vagal control of the heart. This reduction agrees with our previous study in which resistance-trained individuals suffered a decreement of complexity for the same variable (13.9–17.6%) (21). An interesting point in that study is that a significant reduction of complexity was not observed in untrained participant (0.7 to −8.3%) (21). Also, previous studies in recreationally active participants have observed a slight reduction (12.1%) (20) or a nonsignificant reduction in SampEn (2.3–9.7%) (24). Taking all this into account, it is possible that the loss of HRC may be training-status dependent. Furthermore, this response is in contrast to the findings we report for HRV variables, in which training status doesn't seem to be an important factor. This in turn may assist in explaining why HRC has been described as a more sensitive measure (20).

On the other hand, the 2-fold reduction of complexity in our study in comparison with the reductions observed in our previous study using resistance-trained individuals (13.9–17.6%) (21) suggests that other factors besides training status may affect HRC. The relative intensity and total volume could affect the responses. In addition, our whole-body design does not seem to play an important role. Other protocols performing a split or whole-body design have not observed differences between sessions in resistance-trained individuals (21). With this in mind, the free-weight design of the present study may partly explain the differences observed in the loss of complexity. Until now, just one previous study has provided data on the effect of resistance exercise on LZEn. This particular study was a training study in which LZEn increased after training, but returned to resting values after detraining (15). Our study is the first that reports values of this parameter after an acute bout of resistance exercise.

The significant reduction of 63.1% of BRS after the resistance exercise session confirms previous data suggesting that after resistance exercise there is a decrease in the operating point of the baroreflex in young healthy individuals (14,24,27). However, the present study adds to the body of literature, as our methods and participants had not been previously used. This decrease in our study is comparable with previous reports that observed a decrease of about 49.4–59.2% after resistance exercise protocols with a similar design (14,24,27) and slightly higher versus protocols lower in intensity, effort or volume (36.2–37.6%) (24,27). It is known that more demanding protocols cause a higher reduction of BRS in comparison with less demanding protocols (24,27). The reduction in wall deformation and the consequent reduction in the RR interval responsiveness explain the reduction in BRS and the transient increase in cf-PWV (26). This reduced capability is due to a higher sympathetic tone of the central arteries, something observed previously both at rest (5) and after resistance exercise (14).

There are a few limitations of the present study that should be mentioned. Primarily, we didn't have control for the Valsalva maneuver. Our data are only in relation to the aftereffects of an acute bout of resistance exercise, and can't be used to determine the effects during resistance exercise or after a resistance training period. Even while women were tested in their follicular phase, gender differences in cardiovascular control may be a confounding factor.

The transient reductions in parasympathetic control observed after whole-body free-weight resistance exercise should be interpreted as a temporary deleterious effect in resistance-trained individuals. This is because 30 minutes after resistance exercise there is an increased risk of suffer a cardiovascular outcome as sudden cardiac death, due to reductions in parasympathetic activity (1). These reductions should be controlled in individuals with increased cardiovascular risk when resistance exercise is prescribed.

In conclusion, an acute bout of whole-body free-weight resistance exercise produced an increase in arterial stiffness, a reduction in vagal modulation measured by heart rate variability and complexity, and a decrease in baroreflex sensitivity. These data suggest that an acute bout of whole-body free-weight resistance exercises may have a transient deleterious effect on cardiovasculature. Future research should analyze the effect of the different features of the resistance exercise, as differences between free and machine weights, and how these affect to alter the cardiovascular responses.

Practical Applications

Our data demonstrate, for the first time, the cardiovascular effect of a whole-body free-weight resistance exercise session in resistance-trained individuals. This study adds to the literature about how to prescribe resistance exercise through the impact on autonomic modulation when these exercises are completed consecutively. Based on our data, performing these 3 exercises at the given volume (3 sets of 10 repetitions at 75% 1RM) might result in an unfavorable

cardiovascular response. A better option may be to separate these exercises into different training days. Also, this study examines the cardiovascular responses of free-weight resistance exercise, as reductions in vagal control or increases in arterial stiffness, that are associated with postexercise cardiovascular outcomes such as sudden cardiac death, helping to prescribe exercise in diseased individuals. All this together contributes to the body of research to prescribe resistance exercise in a more precise and secure manner.

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